questions sequentially we may arrive at a rational and current approach to therapy of the menopause.

1. Vasomotor instability, atrophy of urogenital structures, osteoporosis and increased emotional lability all characterize the climacteric. An unequivocal relationship between estrogen deficiency and the latter two conditions has not yet been proven.

2. Vasomotor instability occurs in 30 to 75 percent of postmenopausal women and usually persists for 1 to 3 years. This symptom usually responds well to estrogen replacement. Atrophic vaginitis and urethritis, loss of tone of breasts and diminished pelvic support all improve with estrogen replacement. Osteoporosis may be delayed and possibly prevented by estrogen therapy. However, preexisting osteoporosis cannot be reversed. The synergistic effect of exercise and proper diet upon the prevention and limitation of osteoporosis cannot be overemphasized. Indeed, these factors may be more important than estrogen treatment. Improvement in well-being and reduction of emotional lability by estrogen treatment might occur but these symptoms are too intangible to quantitate definitively.

3. A causal relationship between both breast and endometrial malignancy and estrogen treatment of postmenopausal women has long been suspected. Recently, a number of retrospective case control studies have indicated a substantially increased risk for the development of both invasive and noninvasive endometrial cancer among women receiving estrogen therapy. Additionally, a dose-response relationship and a duration-response relationship have been shown. Although retrospective studies suffer from a number of deficiencies, these reports cannot be ignored. Similar studies for breast cancer are less convincing.

With this background, it seems logical that only those menopausal patients suffering from vasomotor symptoms and those with atrophic changes should receive estrogen replacement therapy. Young women in whom bilateral oophorectomy has been done also may be candidates for treatment to prevent osteoporosis. Treatment should be intermittent rather than continuous. The periodic administration of progestins in order to differentiate or slough (or both) the estrogen-stimulated endometrium is worth considering although no studies have shown a protective effect. After one to two years of therapy, administration of estrogens should be discontinued and the need for further treatment assessed. Finally, estrogen treatment is designed to replace endogenous secretion. Pharmacological levels of hormones are to be avoided. A recently modified assay for corticosteroid binding globulin-binding capacity may provide the needed indicator which will allow us to stay within physiological dosages.

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Habitual Abortion

IN A WOMAN who has had three consecutive early spontaneous abortions a comprehensive medical evaluation should be done, followed by specific treatment or reassurance, and continued support before and during subsequent pregnancy. Contraception is recommended until the evaluation is completed. The adequacy of the luteal phase is ascertained by basal body temperature and by endometrial biopsy and serum progesterone studies on the seventh postovulatory day. An apparent defect should be documented in at least one further cycle and the underlying cause further investigated by assessing the adequacy of preovulatory estrogen stimulation.

Deficiency of estrogen effect should be treated by supplementary estrogen from the seventh day until the midluteal phase. Luteal phase insufficiency can be treated by either clomid or gonadotropin stimulation of follicular development, by human chronic gonadotropin stimulation of luteinization or by progesterone substitution during the luteal phase. The effect of treatment should be verified by repeat endometrial biopsy. A culture for T mycoplasma is sent to the virology laboratory and if positive both partners are treated with a ten-day course of doxycycline followed by repeated cultures. The endometrial cavity is evaluated by hysterosalpingogram or, preferably, by hysteroscopy. In addition to identifying uterine anomalies and submucus myomata, which are infrequent causes of early fetal loss, endometrial scarring or denudation resulting from repeated curettage may be directly visualized and treated.

Finally, a banding chromosome analysis is done on both partners. Although more costly,

consistent identification of balanced translocations requires this technique. Genetic counseling should be provided for the 5 to 10 percent of couples for whom etiologic determinants are discovered. Identification of a genetic cause is particularly important, because a subsequent pregnancy may result in the birth of an abnormal viable infant.

Those couples in whom there are negative findings on complete examination or a treatable cause is found should be reassured and offered continuing support in an attempt to avoid perpetuation of failure due to psychosomatic factors.

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CORRECTION

The epitome entitled "Grey Scale Ultrasonographic Differentiation of Medical and Surgical Jaundice," which appeared in the March issue on page 219, was incorrectly credited to Gabriel Wilson, MD. The author was in fact W. Fred Sample, MD.